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Renal Tuberculosis.—A case of tuberculosis of the right kidney diagnosed in 1920 is reported. This patient was followed for nearly twenty years. Tuberculosis of the right kidney was diagnosed subsequent to the characteristic symptoms of cystitis, and urine obtained from the right ureter contained tubercle bacilli on culture and on animal inoculation. The patient declined operation, and within the next twenty years developed tuberculosis of the left epididymis and testis. These were excised without sequelae. Some time later the patient developed bilateral upper lobe tuberculosis. This apparently subsided under general hygienic measures, and still later the patient developed tuberculosis of the prostate, which subsided in spite of the fact that no special treatment was carried out. At seventy-one, the patient is still alive and well. Initially, he received some months rest and tuberculin treatment for the renal tuberculosis. The question is raised whether the renal tuberculosis really healed, or remained encapsulated so that no further bacilluria was noted. The haematogenous seeding to the other organs may have had its origin in an encapsulated kidney focus, but on the other hand a bronchopulmonary lymph node may have been the focus which infected the kidney initially and later on the other organs. The author advises nephrectomy in all cases of unilateral renal tuberculosis.—*Zur Frage der konservativen Heilung der Nierentuberkulose, W. Hofman, Schweiz. med. Wchnschr., February 7, 1942, 72: 154.*—(*H. M.*)

Intestinal Tuberculosis.—Roentgenograms allow the diagnosis of tuberculous enteritis with a great degree of accuracy. However, a clinical study alone will often enable one to diagnose the presence of intestinal tuberculosis. The development of intestinal tuberculosis depends upon the activity and progression of the systemic disease. This study was undertaken to provide a correlation between the occurrence of intestinal tuberculosis and the presence of an elevated temperature in patients with chronic pulmonary tuberculosis. The patients studied were divided into three groups: (*1*) those that were afebrile; (*2*) those that were subfebrile with temperature elevations to 101°F. by rectum; and (*3*) the febrile ones, those whose temperatures were over 101°F. by rectum. The elevation of temperature is an index of the activity of pulmonary tuberculosis only if there is no other cause. There is no correlation between temperature elevation and the presence of intestinal disease, unless the fever has been persistent for more than two months. Sex, color and duration of the disease are not significant in the development of intestinal tuberculosis. The presence of active pulmonary tuberculosis with persistent elevation of temperature is the most constant determining factor in the development of intestinal tuberculosis. About 95 per cent of those patients with fever which persisted for more than two months were found to have intestinal tuberculosis at post-mortem examination.—*The Importance of Fever as a Diagnostic Sign of*

Intestinal Involvement in Chronic Pulmonary Tuberculosis, I. G. Tchertkoff & H. Green, Quart. Bull. Sea View Hosp., April, 1942, 7: 230.—(A. A. E.)

Demonstration of Tubercle Bacilli.—Various techniques for the demonstration of tubercle bacilli are compared. The conventional Ziehl-Neelsen method has been modified by Osol in 1927. He uses the usual carbol-fuchsin stain, decolorizes with 5 per cent sulfuric acid and, after rinsing with water, he rinses the slide with 10 per cent sodium sulfite solution or exposes it for ten to fifteen seconds to ammonia. If the preparation is viewed in the darkfield, the tubercle bacilli stand out very clearly. Comparative results show that this method is much superior to the usual Ziehl-Neelsen technique, although the results of the latter can be improved when the slides are viewed in the darkfield. Out of 100 preparations found positive by the Osol darkfield method, 47 were found positive by the Ziehl-Neelsen method, and 69 were positive when the Ziehl-Neelsen preparations were viewed in the darkfield. The method described is as satisfactory as the fluorescent technique. In addition it is cheaper and simpler.—*Der Nachweis von Tuberkelbazillen im direkten Ausstrich, gefärbt nach Osol, betrachtet im Dunkelfeld, als Methode der Wahl, H. Egli, Schweiz. med. Wchnschr., February 7, 1942, 72: 166.—(H. M.)*

Demonstration of Tubercle Bacilli by Fluorescence.—In view of the difficulties encountered with the Ziehl-Neelsen method, the introduction of a cold stain, followed simply by a decolorizing solution, is appealing in itself. Such a procedure is the fluorescent technique. Sputum submitted to the laboratory arrives in cresol; it is autoclaved and then centrifuged. A total of 1,221 specimens were examined for tubercle bacilli with the fluorescent and the Ziehl-Neelsen techniques. Of these, 1,123 were specimens concentrated by cresol, autoclaving and centrifuging. The fluorescence technique for the examination of tubercle bacilli was more sensitive than the Ziehl-Neelsen staining method, increasing positives by 1 per cent, although the use of cresol in concentration produced fluorescence which interfered with the examination of the specimens. Because of the larger areas covered and the ease of examination, the time required to examine the smears was reduced by one-fourth to one-third. The disadvantages of a monocular microscope, the high degree of miscellaneous fluorescence, and the possible fatigue were counteracted by the advantages of larger microscopic fields, simplicity of technique, rapidity of procedure and increase in number of positives. The simplicity of the technique permits a larger number of specimens to be handled in a shorter period of time.—*Fluorescent Staining Technique for Detection of Acid-Fast Bacilli, H. E. Lind & H. J. Shaughnessy, J. Lab. & Clin. Med., January, 1942, 27: 531.—(F. G. P.)*

Sputum Examination.—In order to evaluate various methods for the examination of sputum for the presence of tubercle bacilli, 3,332 sputum specimens were examined according to several different procedures. By direct smear, 1,081 specimens were examined and of these 703 contained tubercle bacilli. The 378 negative specimens were then studied by the Greenfield-Anderson concentration method and 74 (19.6 per cent) were found to contain tubercle bacilli. A second series of 1,000 specimens examined by direct smear revealed 614 positive specimens. Further examination of the negative specimens by the sodium hydroxide concentration method disclosed 100 (29.5 per cent) more positive samples. A third series of 821 specimens examined by direct smear contained 476 positive specimens. The negative specimens in this series were then digested and concentrated by the Tergitol-Javelle Water method and 151 (43.7 per cent) were shown to contain acid-fast bacilli. This latter method therefore not only disclosed more positive specimens, but required very much less of the laboratory technician's time

to prepare and stain the smears. This method of preparation and staining also resulted in a more brilliant appearance of the organisms. The efficiency of the Tergitol-Javelle Water method is due largely to the digestive properties of the reagent which produces a better concentration of the material after centrifuging.—*Sputum Examination. A Comparative Study of Several Concentration Methods, P. Schain, S. Magdalin & A. Russo, Quart. Bull. Sea View Hosp., January, 1942, 7: 99.*—(*A. A. E.*)

Postprimary Tuberculous Complexes.—The author's observations have led him to believe that the classical complex of parenchymal and lymph node focus is frequently erroneously referred to as "primary complex." He prefers the term initial complex (Startkomplex) for these characteristic lesions, consisting of a small subpleural focus and swollen caseous tracheobronchial and bronchopulmonary nodes. From autopsy material he has learned that frequently two so-called primary complexes of different ages are found in the same individual. He believes that the entire cycle of infection, allergy, healing and immunity takes three to five years. After this time the organism may be ready for another infection, which again takes the characteristic shape of the so-called primary complex. His autopsy material includes the case of an eleven year old girl who showed one completely healed primary focus and progressive tuberculosis emanating in a postprimary focus. There were very large caseous nodes. Adults are also subject to progressive postprimary tuberculosis. The lymph node lesions are usually less extensive. The author believes that the postprimary complex is caused by exogenous reinfection in a normergic individual.—*Ueber tuberkulöse postprimäre Startkomplexe, P. Schwartz, Schweiz. med. Wchnschr., February 7, 1942, 72: 141.*—(*H. M.*)

Malformations of Lung.—A review of the various types of structural anomalies of the lung is presented. The occurrence of such anomalies may be favored by the relative newness of the lung from a phylogenetic standpoint and by its enormous growth in embryo before becoming functionally active. Prevailing opinion indicates that after evagination from the foregut the bronchial tree develops by arborization and downgrowth, and this genesis is important in explaining most instances of congenital abnormality. Inhibition or aberration of this branching may result in corresponding defects or excesses in the associated pulmonary tissue. Thus, arrest of bronchial development at successively descending points may result in the absence of both lungs, of one lung or in lobar anomalies such as fusion of the right upper and middle lobes if the eparterial bronchus fails to develop. Anomalies of the main bronchi which later develop from the stem bronchi, and in the development of the secondary bronchi, are only infrequently observed since they are not reflected in the gross lung configuration. Cystic disease of the lung, often called congenital bronchiectasis, occurs with comparative frequency. The cysts may be numerous and small (honey-comb lung) or few and large (pneumatoceles) and are in most instances dilated bronchial branches. They may result not only from unknown developmental factors, but also from a number of diseases acquired prenatally or postnatally. The latter idea is favored, too, by the fact that pulmonary differentiation has been shown to continue for some years after birth. The cysts might be formed by continued growth of living cells of an air path while the organizing influences favoring branching and folding are inhibited by the failure of adjacent pulmonary tissue to assume its proper volume, or by symmetrical narrowing with stagnation of secretion or air. Various other aetiological factors have been suggested, including primary agenesis of finer ducts and alveoli, neoplastic hyperplasia of the bronchial tree, congenital deficiency of elastic tissue, foetal (especially syphilitic) bronchopneumonia, lobular pneumonia in childhood and others. Another group of pulmonary anomalies in-

cludes supernumerary formations. Of these, the azygos lobe is a deformity not inherent in the pulmonary anlage, it is, in reality, a portion of the upper lobe split off by the azygos vein deviating too far laterally and lifting a fold of pleura from the posterior thoracic wall. Supernumerary formations due to developmental anomalies include the rare tracheal lobe, sometimes confused with the azygos lobe due to position, and apparently formed by a bronchus arising directly from the trachea, and, in addition, the not quite so rare tracheal diverticula, representing potential tracheal lobes. These are believed most likely to be supernumerary right apical main bronchi. A left apical lobe may be present, possibly due to an anomalous left eparterial bronchus; accessory lobe formation may also occur around the infracardiac bronchus, more frequently on the right where this branch arises from the main stem bronchus. These tributary accessory lobes must be differentiated from the "accessory lungs," masses of pulmonary tissue usually occurring at the left lung base and consisting of lung tissue developed to varying degrees with bronchi having no communication with the normal lungs. In a few instances, such accessory formations have been found below the diaphragm. Theories of formation include sequestration of a portion of a large bronchus in the course of development, the occurrence of adhesions between the lung blastema and the coelom mesothelium, with resultant sequestration of a portion, and, finally, the formation of a second primitive respiratory tract from the digestive tract. This last theory is not generally accepted as an origin for lower accessory lungs; two instances of true ectopic lungs due to displaced anlage of the stem bronchi have, however, been described.—*Malformation of the Lung, G. E. Gruenfeld & S. H. Gray, Arch. Path., March, 1941, 31: 392.*—*(D. G. F.)*

BCG Vaccination.—A drop of BCG was placed on the outer aspect of the alcohol cleansed arm and 35 to 45 punctures were made over an area 2 by 1.5 cm. This method seems to be superior to the conventional intracutaneous administration of the BCG vaccine because: (*1*) the local lesion is more discrete and leaves only depigmentations and no scars; (*2*) although regional lymphadenitis regularly develops, the lymph nodes do not break down; (*3*) allergy develops earlier and is as lasting as with the intracutaneous method. From supposedly nontuberculous environment, 484 newborns between the ages of three to seven days were vaccinated with BCG by the multiple puncture method. They were periodically retested with 0.1 to 0.5 mg. OT intracutaneously. By the time they became eleven to thirteen days old 14 per cent developed a positive Mantoux test. Between the ages of twenty-three to thirty-one days 97 per cent of them reacted to tuberculin. The positive Mantoux test persisted in 79 per cent of the cases as late as thirty-four to thirty-nine months of age. There were 613 nonvaccinated control infants also similarly observed and tuberculin tested. Among these between the ages of thirty-four to thirty-nine months 19 per cent became tuberculin positive and one of them died of tuberculosis. Among the 484 infants vaccinated by the multiple puncture method and among the 191 infants who received BCG by the intracutaneous method none died of tuberculosis. The percentage of reactors in newborn infants was not appreciably altered if 3.5 to 9 mg. vaccine diluted in one-fourth strength of Sauton medium containing 1.5–50 per cent glycerine was used. The authors give a very plausible histological explanation for the precocity of the appearance of tuberculin reactivity following the multiple puncture. As the majority of phthisiologists consider allergy to tuberculin a concomitant of relative immunity against tuberculosis, it is of practical importance to find for administering BCG a method which shortens the preallergic period after immunization. Using the multiple puncture method this preallergic period is less than one month. The authors express their opinion that it is only fair to expect as much and not more from the BCG than from other forms of

vaccination. This means that the patients shall be uninfected before vaccination and shall not be exposed to tuberculosis after vaccination until a positive tuberculin test develops. By these precautions Wallgren was able to protect all his 228 BCG vaccinated contacts from developing tuberculosis.—*Clinical Studies on the Harmlessness of and Tuberculin Reactivity following BCG Vaccination by the Multiple Puncture Method, S. R. Rosenthal & I. S. Neiman, J. Pediat., July, 1941, 19: 16.*—(*C. K.*)

Promin in Tuberculosis.—Promin, when given intracutaneously in doses of 160 mg. three times daily to guinea pigs, exerted a retarding effect on tuberculosis, but did not cure the disease. Promin, when added to Sauton's medium in concentration of forty mg. per cent, inhibited the growth of tubercle bacilli for about thirty days, after which time the organisms began to grow.—*Promin in Treatment of Experimental Tuberculosis, M. M. Steinbach & C. J. Duca, Proc. Soc. Exper. Biol. & Med., March, 1942, 49: 460.*—(*F. B. S.*)

Proteinases in Experimental Tuberculosis.—Cellular proteinases (cathepsins) have been demonstrated in the polymorphonuclear leucocytes, monocytes and epithelioid cells of rabbits. A study was made of the behavior of these proteinases in the various organs of rabbits experimentally reinfected with virulent bovine tubercle bacilli after having been previously immunized with a strain of low virulence (R-1). Infection was carried out by the agar focus technique, and only those animals with strong positive tuberculin reactions were reinfected. The rabbits were killed by exsanguination two weeks after reinfection and the catheptic activity of the spleens, livers, kidneys and lungs was measured in terms of micrograms of tyrosine liberated from haemoglobin by the aqueous extract of 8 mg. of frozen pulverized tissue. The spleens showed a marked decrease in cathepsin (242 to 363 micrograms of liberated tyrosine as compared with a control range of 411 to 545 micrograms); the livers also showed marked decreases (76 to 117 as compared with a control range of 127 to 225). The kidneys showed less significant decreases and the changes in the lungs were negligible. Tissues of rabbits reinfected with organisms of low virulence showed no appreciable reduction in cathepsin. The largest decreases occurred in those organs which Lurie demonstrated to be most efficient in destroying virulent tubercle bacilli injected intravenously. In vitro experiments showed that tuberculophosphatide obtained from virulent bacilli inhibited leucocytic and tissue cathepsins, whereas tuberculin, purified protein derivative and tuberculo-polysaccharide had no significant effect. It is possible that the phosphatide liberated by the destruction of tubercle bacilli may be responsible for the inhibition of the catheptic activity in the spleen and liver.—*Studies on Inflammation: I V. Behavior of Cellular Proteinases in Experimental Tuberculosis in Rabbits, C. Weiss, Arch. Path., February, 1942, 33: 182.*—(*D. G. F.*)

Chemotaxis of Bacterial Fractions.—Tuberculophosphatide obtained from the H-27 strain of human tubercle bacilli, and the tuberculo-polysaccharide from the A-10 strain were tested for chemotropic effect on human and rabbit polymorphonuclear leucocytes. The test was performed by placing a small amount of test substance on a clean slide, covering with a cover slip on which had previously been spread a drop of rabbit plasma-leucocyte suspension or human blood, and examining the preparation in a warm chamber under the microscope. The test substance thus formed a target by which the leucocytes were attracted or repelled. When powdered phosphatide was used as a target it was found to be toxic; the leucocytes near it showed a decrease or cessation of motility, many of them subsequently dying. A target of inert titanium dioxide moistened with a dilute solution of phosphatide had a weak attractive effect. On the other hand, kaolin moistened with a solution of polysaccharide

weakly repelled the cells.—*Chemotactic Properties of Tuberculophosphatide and Tuberculopolysaccharide, W. B. Wartman & E. S. Ingraham, Jr., Arch. Path., June, 1940, 29: 773.*—(*D. G. F.*)

Collodion-agglutination in Tuberculosis.—After several of the causes for false positive reactions in this method were eliminated, only about 4 per cent of human tuberculous sera gave specific positive reactions.—*Collodion-agglutination Test in Human Tuberculosis, R. P. Morris, Proc. Soc. Exper. Biol. & Med., May, 1942, 50: 172.*—(*F. B. S.*)

Sensitization by Means of Adjuvants.—When guinea pigs receive an injection of horse serum combined with a lanolin-like substance (Aquaphor) and killed tubercle bacilli suspended in oil, sensitization to horse serum is similar to that seen in guinea pigs injected with living tubercle bacilli and horse serum; that is, the reaction to the intracutaneous injection of horse serum lasts longer than forty-eight hours and may lead to necrosis. The duration of intense sensitization is remarkably long and precipitin titres are higher than when guinea pigs are immunized without the aid of adjuvants.—*Sensitization to Horse Serum by Means of Adjuvants, J. Freund & Katharine McDermott, Proc. Soc. Exper. Biol. & Med., April, 1942, 49: 548.*—(*F. B. S.*)

Collodion-particle Agglutination.—Comparison of the delicacy of the precipitin test and the collodion-particle agglutination test was made with the use of one lot (5807) of antiserum from horses sensitized to defatted tubercle bacilli. The results indicated that the latter test was more delicate than the precipitin test, particularly for the detection of the polysaccharide antibody which this serum contained.—*Collodion-particle Agglutinative Tests in Detection of Antigen Derived from Tubercle Bacilli, J. T. Riordan, Proc. Soc. Exper. Biol. & Med., April, 1942, 49: 622.*—(*F. B. S.*)

Immunity and Tuberculin Reaction.—The frequency of development of tuberculosis is compared in 1320 student nurses who reacted to tuberculin on entering training in 4 New York City Hospitals and 910 students who were classified as non-reactors (half of them had been negative to both strengths of tuberculin and half had been tested with the first dose only). Six students or less than 0.5 per cent of the first group developed tuberculous lesions, while 34 or 3.7 per cent of the non-reactors developed lesions. The author concludes that a positive tuberculin reaction may be considered an indication of immunity to reinfection, comparable to the immunity to exogenous infection conferred by a primary syphilitic lesion. The author states that "doctors, nurses, attendants and students who are tuberculin-positive need not fear tuberculosis because of their work. A tuberculosis patient is not a source of infection for adults of his family who are tuberculin-positive reactors."—*Immunity and Positive Tuberculin Reaction, L. Brahdy, Am. J. Pub. Health, October, 1941, 31: 1040.*—(*H. L. I.*)

Pulmono-cardiac Failure.—Pulmono-cardiac failure refers to the fatal consequences arising from the distortion of the thoracic organs in some patients with extreme deformity of the spine. Among 6,700 autopsies since 1925, 5 instances of this condition were observed. It is generally believed that persons with such deformity tend to die in early adult life. However, in this series the average age at death was fifty-three years. All the patients were men. Scoliosis is usually associated with kyphosis but in 2 of these patients, one due to injury and one to tuberculosis, kyphosis alone was present. Dyspnoea was the major symptom in all cases. Mild symptoms were usually present for several years or even as far back as the patient could remember. The length of time from the onset of the more acute manifestations to death was relatively short, usually several months, and rapid deterioration usually took place in the last

few days. Four of the patients gave a history of intermittent swelling of the feet. Cyanosis was present in all patients, involving chiefly the head and neck. In 2 patients, the localized distribution of the cyanosis, together with the dilatation of the veins, suggested a superior mediastinal mass. No cardiac murmurs or abnormalities were detected. The only consistent signs of cardiac failure were basal râles. In 4 patients, there was marked right ventricular hypertrophy as determined by measurement of the right ventricular wall. Dilatation of the right side of the heart was present in 4 cases. Hypertrophy of the left ventricle was not present in any case. There were no valvular lesions. In all patients, the lungs were small. Atherosclerosis was moderate to severe in the larger branches of the pulmonary artery in 4 of the 5 cases. Pulmonary infection was not a factor in any case. There were no significant microscopical abnormalities in the hearts or lungs. The mechanism of production of failure in this type of case is not definitely settled. Most evidence indicates that the diminution in the size of the lungs and the reduction in the vital capacity with deficient oxygenation of the blood throw an increasing strain on the right side of the heart. In the absence of severe pulmonary arteriolar sclerosis, the apparent pulmonary hypertension with failure of the right side of the heart must be explained by the concept of a purely mechanical effect on respiratory function in these patients with severe spinal deformity.—*Pulmonocardiac Failure as a Result of Spinal Deformity, A. J. Kerwin, Arch. Int. Med., April, 1942, 69: 560.*—(*H. R. N.*)

Pulmonary Infarction.—Tight ligation of a lobe of lung tissue in 5 guinea pigs produced anaemic infarcts in all cases after twenty-four hours. Loose ligation of a lobe produced haemorrhagic infarcts in 7 out of 10 guinea pigs, the remainder showing only hyperaemia and atelectasis. Ligation of a portion of the lungs of 25 rats against the chest wall by introducing a suture into the thorax, produced haemorrhagic infarcts in 15; no anaemic infarcts could be produced in this way. On the other hand a true haemorrhagic infarction of the kidney could not be produced by loose ligation of the renal vessels or of the renal substance, though anaemic infarcts could be produced with ease. The haemorrhagic infarcts observed for more than twenty-four hours in a few animals in no case developed into anaemic infarcts. In most instances occlusion of the pulmonary artery at any point does not produce severe changes in the pulmonary tissue supplied because of the collateral circulation through the bronchial arteries; anaemic infarcts have been described but are rare. If blood continues to enter the tissue and remains under high pressure as in cases of venous congestion, haemorrhage follows and a haemorrhagic infarct may be considered an extreme degree of passive congestion. Increased venous pressure appears to be the only reason that haemorrhagic rather than anaemic infarcts occur in the lungs but both must be considered exceptional occurrences.—*Pulmonary Embolism and Infarction, L. Loeffler, Arch. Path., January, 1941, 31: 93.*—(*D. G. F.*)

Pulmonary Syphilis.—A man of thirty-seven years with a history of secondary syphilitic lesions eight years previously complained of respiratory distress, characterized as oppression, following an upper respiratory infection. This single symptom persisted despite two months of bed-rest. Sputum was repeatedly negative for tubercle bacilli. X-ray examinations showed a progressive infiltration in the left lower lung field. Five months after onset there was an extensive area of infiltration adjoining the heart shadow in the lower part of the left lung. Wassermann and Kahn reactions were strongly positive. A course of therapy including iodide, bismuth and neosalvarsan resulted in complete subjective improvement within two months. X-ray films showed marked clearing of the infiltration. The bronchi to the lower part of the left lung appeared thickened

and a rounded shadow, suggesting a gumma, was still visible. Five months after the beginning of treatment, an X-ray film showed complete clearing except for increased bronchovascular markings. The frequency of syphilis of the lung is underestimated. At autopsy lesions are not always gummatous. The only evidence of syphilis may be small areas of scarring which may easily be overlooked. Syphilis rarely affects the pulmonary arteries but has a great affinity for the bronchi. Here it sets up lesions of a complex and intense character, which may be distinguished from tuberculosis by their appearance alone. Careful study of the bronchi is essential since marked damage may occur without alteration of the parenchyma. The lesions of pulmonary syphilis occur in the form of multiple foci most commonly in relation with the bronchi rather than the blood vessels. There are always marked changes in the lymphatics. These foci, which may be bronchopneumonic or interstitial, may develop into caseogummatous or productive lesions, which eventually lead to fibrotic scars resistant to anthracosis and calcification. The tendency to caseation is very limited and the caseous areas tend to resolve rather promptly without spreading. About each syphilitic focus marked connective tissue proliferation develops. It may extend rather far from the actual focus. Such extensive fibrosis without caseation is not seen in tuberculosis. A characteristic of pulmonary syphilis, where it extends to the pleura, is localized and dense fibrosis of the pleura with marked tendency to contraction. The clinical and radiological picture is variable.—*Sífilis terciaria del pulmón, H. Orrego Puelma, Ap. respir. y tuberc., July–December, 1941, 6: 246.*—(*H. B. S.*)

Bronchiectasis Due to Syphilis.—A number of writers hold that both congenital and acquired syphilis play an important rôle in the production of bronchiectasis. The frequency of haemoptysis in bronchiectasis is referable to the presence in the submucosa of the affected bronchi of fresh connective tissue with many large, dilated, thin-walled capillaries which rupture under slight pressure. A man of twenty-five years acquired a primary syphilitic lesion in 1937. In 1938, while still receiving specific therapy, he began to notice a tickling sensation in the throat, cough and blood-streaked sputum. He did not stop work, however, until one year later when he suffered a series of severe haemoptyses which resulted in anaemia and asthenia. He was still receiving specific therapy. Aside from pallor, examination showed only harsh breath sounds over the lower third of the left lung. Wassermann and Kahn tests were negative. X-ray study with lipiodol revealed cylindrical bronchiectases of the left lower lobe. Because of the severe haemorrhages, localized involvement, and failure of specific therapy to affect the pulmonary lesion, lobectomy was performed. The patient recovered. In the extirpated lobe were found several grayish, firm nodular lesions embedded in normal parenchyma. The bronchi showed slight cylindrical dilatation and inflammatory thickening of the walls. Histological study revealed multiple parenchymal areas of interstitial mononuclear infiltrations and fibrosis. These tended to be perivascular and peribronchial. In the bronchi, the infiltrations were situated primarily in the mucosa, sometimes localized and sometimes diffuse. The arteries were more involved than the veins by the mononuclear infiltrations. In some of the infiltrations were areas of haemorrhage. A diagnosis of sclerogummatous syphilis was made on both gross and histological findings.—*Bronchiectasia hemoptóica por sífilis de pulmón. Curación por lobectomía, O. P. Aguilar & C. A. Bancalari, Arch. argent. de tisiol., October–December, 1941, 17: 464.*—(*H. B. S.*)

Bronchiectasis Associated with Monilia.—A case of bronchiectasis in conjunction with *Monilia albicans* is reported. The authors are of the opinion that mycotic infection of the respiratory tract is of more frequent occurrence than is generally believed, and that careful search of cases simulating pulmonary tuberculosis in the absence of tubercle

bacilli in the sputum will reveal this condition, especially where the lesion is of the basal type. However, the authors do not wish to leave the impression that they believe that monilia was in any way responsible for the bronchiectasis; but rather that the bronchiectasis offered a favorable soil for the growth of the fungus once it had reached the lung. Another point worthy of note is that bronchiectasis and moniliasis, both preëminently basal affections of the lung, individually closely simulate basal pulmonary tuberculosis. The combination of the two diseases certainly more than doubles the resemblance. The fact that the specimens were not obtained directly with the bronchoscope need not militate against the results as all possible means were taken to eliminate oral contaminations. If this failure is a fault, then it is one shared by all cases found in the literature.—*Bronchiectasis Associated with Monilia Simulating Pulmonary Tuberculosis, A Clinical-Pathological Study, E. R. Wiese & E. W. Bixby, J. Lab. & Clin. Med., January, 1941, 26: 624.*—(*F. G. P.*)

Reversible Bronchiectasis.—The reversibility of bronchiectasis in the early stages of the disease is an established fact. A case is reported in which reversibility was observed. At the age of thirteen the patient first complained of fatigue, weakness and weight loss following a tonsillectomy. After a few months, foul tasting sputum indicated putrid bronchitis. Shortly afterwards roentgenograms showed consolidation in the left lung base. A year after the tonsillectomy lipiodol showed cylindrical bronchiectasis in the left lower lobe, the lingula of the left upper lobe, and right middle lobe. During the next several months, all objective and subjective signs of the disease, including cough and expectoration of foul sputum disappeared. Roentgenograms nine months after the original ones showed no definite consolidation of the left base. The bronchogram showed no change in the bronchiectasis of the right middle lobe and lingula, but the bronchial tree of the left lower lobe appeared normal. Later, however, the condition grew worse and bronchography again showed very extensive cylindrical dilatation of the bronchi in the left lower lobe with no filling of the small bronchial twigs. After lobectomy the lingula was found to be contracted but the lower lobe was not appreciably shrunken. Pathological study showed bronchial dilatation without bronchial wall thickening. The lung parenchyma showed chronic pneumonitis, that is, some inflammation with atelectasis.—*Reversible Bronchiectasis, F. G. Fleischner, Am. J. Roentgenol., August, 1941, 46: 166.*—(*W. H. M.*)

Roentgenology of Influenzal Pneumonitis.—There is no satisfactory classification of pneumonias occurring in influenza. The lesions show a marked variability and have been variously classified by different writers. Several types are recognizable: (*1*) lobular consolidation: this often implicates a large part of the substance of the lung (pseudolobar), (*2*) peribronchiolar consolidation: the inflammation is limited to the alveoli and alveolar ducts immediately about bronchioles; small nodules are widely scattered in the substance of the lung (referred to as miliary bronchopneumonia or minute acinous foci), (*3*) peribronchial consolidation: any one of alveoli encircling small bronchi is affected. In all forms suppuration and necrosis are common; (*4*) unresolved or organizing bronchopneumonia may occur in any of the foregoing conditions, so that fibrous tissue may be formed about bronchi and blood vessels, in interalveolar walls and in interlobular septa. The lesion is invariably accompanied by purulent bronchitis and usually by bronchiectasis. The polymorphous roentgenological appearance of the lungs affected by influenzal pneumonia makes it very difficult to differentiate this condition from other diseases and especially from pulmonary tuberculosis. The localization of the shadows offers only general, but no definite differential characteristics. Nor is the nature of the shadow (homogeneous, discrete or nodular appearance of the shadow) of decisive value. The clinical picture, and

above all, observation of the whole course of the disease will frequently permit a correct diagnosis. The most important and significant roentgenological indicator of pneumonia due to influenza in such cases is the rapid changes of the original shadow. Seven cases are reported. Clinically some of the cases presented a conspicuous absence of a history of an acute onset. The complaints resembled those of tuberculous patients—general weakness, moderate elevation of temperature, anorexia, occasional dry cough, night sweats, but no chest pain. In other cases, patients developed acute pulmonary symptoms (cough, haemoptysis and high fever), following a prolonged general malaise. The roentgenograms of these patients showed a pulmonary infiltration, which varied in individual cases in extent and localization. On the whole, the infiltration had a tendency to localize in the upper lung fields in the subclavicular region. Roentgenograms of the chest presented shadows of homogeneous density, with somewhat ill defined borders, near the lobar margin of the upper lobe or marked peribronchial thickening with poorly defined contours, extending into the periphery. The greatest density of these shadows was noted in the hilum. In some of these cases a mistaken diagnosis of pulmonary tuberculosis was made, based on the roentgenological findings. It is considered of paramount importance to have these cases examined at short intervals in order to follow the changes occurring in the involved parenchyma. Cases of influenzal pneumonia are characterized by great changeability of the infiltration, whereas pulmonary tuberculosis is more stationary, or if not treated may become progressively worse.—*Roentgenological Consideration of Influenzal Pneumonitis, A. J. Ackermann, Am. J. Roentgenol., August, 1941, 46: 175.*—(*W. H. M.*)

Fibrinous Bronchitis.—About 100 cases are reported in the literature. The author differentiates between acute and chronic types of this disease. The acute type is characterized by sudden onset of cough, dyspnoea and expectoration of bronchial casts. The course is severe and the mortality is around 50 per cent. Fatal cases frequently end with massive pulmonary infection, atelectasis and cardiac failure. Chronic fibrinous bronchitis is seldom fatal but the patient may remain an invalid for many years. These patients are subject to frequently recurring attacks of fever, cough, dyspnoea, associated with expectoration of bronchial casts. Pneumonic consolidations during the attacks are observed. An attempt has been made to separate the disease into primary and secondary forms. In the latter the disease may be a sequela of pneumonia, bronchitis, tuberculosis, etc. Genuine fibrinous bronchitis supposedly is not associated with other disease of the respiratory tract. This differentiation is believed to be impossible, because in most cases observed, an inflammation of the tracheobronchial tree is believed to precede the onset of the disease. The disease is frequently associated with diseases of the vegetative nervous system or with endocrine disturbances. The rôle of these factors appears to be predisposing in nature. The author adds one case to the literature which he has observed over ten years. His patient was a woman of forty-four who also had hyperthyroidism. He was able to treat the patient successfully with pneumothorax. She remained well and free from attacks over two years, and apparently a beneficial influence on the endocrine disturbance was also achieved. The mode of action of pneumothorax therapy in this disease is ill understood. —*Über Bronchitis plastica, S. Seiler, Schweiz. med. Wchnschr., January 24, 1942, 72: 86.*—(*H. M.*)

Resection of Pulmonary Plexus for Asthma.—The results following resection of the posterior pulmonary plexus for intractable asthma are reported. The operation is based on the fact that the extrinsic nerve supply of the lungs consists of two entirely independent tracts; only by resection can this supply be completely interrupted. The nerve tracts consist of efferent fibres of both the vagus and the

sympathetic bronchomotor tracts from the same and contralateral sides, together with the sensory or afferent fibres emerging from the lung. Bilateral interruption is therefore necessary to obtain what might be described as the expected physiological results, the prevention of constriction of the bronchi and of excessive secretion from the bronchial glands. Twenty-one patients were treated by resection of the pulmonary plexus. All of the patients were critically ill with an intractable disease; medical procedures failed to give even transient relief. Without resort to drastic treatment the outcome of the disease was as obvious as in any case of advanced malignant disease. The asthma, in every instance, was of the intrinsic type, the result of chronic respiratory infection and the subsequent infection of the pulmonary system. Allergy from extrinsic factors apparently played little part in the production of the asthmatic paroxysms. Of these patients, 8 derived striking benefit, 9 died as a result of, or subsequent to, the operation, and 4, who failed to improve following operation are still suffering from severe bronchial asthma. Apparently these 4 patients should not have been subject to this radical procedure. Although they showed signs and symptoms of violent *status asthmaticus*, their attacks invariably followed emotional disturbances or mental strain. They were fortunately none the worse following the operation. The 21 patients had been ill from one to twenty-eight years. The duration of the disease had no apparent effect on the therapeutic success of the operative procedure. One patient, thirty-nine years of age, had suffered with asthma for twenty years; since the operation eight years ago she has never had an attack of asthma. The ages of the patients seemed to have no relation to the outcome; the youngest was twenty-five years of age and the oldest fifty-five. With one exception, none of the patients derived immediate relief from the operation. It apparently takes some time for the inspissated mucus to be eliminated. The seriousness of the operation should not be minimized. If it is quite definitely established that no other method of therapy, or change of environment, is likely to give relief to the malignant asthmatic, then one would seem justified in advising denervation of the lungs.—*Further Observations on the Treatment of Intractable Bronchial Asthma by Bilateral Resection of the Pulmonary Plexus, L. N. Gay & W. M. Rienhoff, Bull. Johns Hopkins Hosp., April, 1942, 70: 386.*—(*J. S. W.*)

Coccidioidomycosis.—This disease caused by the inhalation of *Coccidioides immitis* is characterized by symptoms closely parallel to those of influenza or bronchopneumonia. The onset is sudden with headache, weakness, anorexia, nausea, dry cough, pleuritic pain and occasionally chills, night sweats, fever and malaise. The second stage is characterized by the appearance of erythema nodosum, arthritis and conjunctivitis. The diagnosis is made by finding the typical spherules in the sputum, a coccidioidin reaction, positive agglutination tests, animal innoculation, leukocytosis and eosinophilia. Roentgen findings are those of a bronchopneumonia indistinguishable from that found in epituberculosis. Pleurisy and cavitation may occur. Among the cases reported multiple lesions were present from the start in the severe cases, while in the milder type solitary or a few lesions were present. In the severe form the multiple lesions may be progressive, and new lesions may appear, some of which may go on to cavity formation. The latter seem to form and close quite rapidly, although in one case, a small cavity remained for over a year. The lesions are usually homogeneous in density and many are distinctly nodular and have a peripheral posterior distribution. No calcification can be demonstrated. The condition is most easily confused with primary tuberculosis or pneumonia.—*Acute Coccidioidomycosis, R. A. Powers & D. J. Starks, Radiology, October, 1941, 37: 448.*—(*G. F. M.*)

Silicosis.—Simple silicosis is not a disease, is not disabling and is not a cause of death. It does not predispose to tuberculosis. Diversity of opinion on the part of medical

witnesses is due to an inadequate understanding of what simple silicosis really is, the influence of disease upon it, the failure to realize that the incidence of infection is no greater in silicotics than others and failure to realize that simple silicosis is not a disabling condition. Sympathy for the plaintiff without determining the cause of his diability is common. Simple silicosis is rarely seen in the hospital or clinic for it does not require the attention of a physician. In the prenodular stage the appearance is that of enlarged hila with a relative increase in the density and size of the normal linear markings which nowhere touch the periphery. There is no parenchymal involvement, although there may be a faint haziness. In this stage, also, small discrete spots are often apparent close to the hila and linear markings. The nodules rarely increase in number, but merely in size so that in later years they appear to occupy more and more space in the lungs. As the condition develops there is some emphysema present or the nodulation appears to overshadow the linear increase. The apices are never involved and the costophrenic angles are clear. There is no change in the size or shape of the heart and any deviation from normal in the appearance of the thoracic organs, except the discrete nodulation, is *prima facie* evidence of disease and is in no way associated with simple silicosis. Tuberculous infection occurs no more frequently among silicotics than in the non-silicotic contiguous population.—*Silicosis, G. Clement, Radiology, October, 1941, 37: 407.*—(*G. F. M.*)

Pulmonary Concussion.—Experimental and theoretical considerations have now established that blast injuries of lungs are produced by the high pressure waves of the blast acting for a short period. It is due to a sudden diffuse compression of the lungs through the chest and abdominal walls and is typically unaccompanied by obvious injuries of the thoracic wall. A study of 94 necropsies made during the last three years on persons dying through road accidents, revealed three who had lung lesions comparable to those of the bomb cases. These 3 were children aged four, seven and eight. The best is a girl of four who had been run over by a motor-lorry. Her lungs showed multiple haemorrhages, which all appeared on the pleural surface; they were most numerous in the parts of the lung which enter the phrenico-costal sinus of the pleura and posteriorly. It is noteworthy that all three cases were in children, probably due to their flexible chest walls. Eight cases from bombed houses are described. They all showed essentially the same microscopical features. Two main types are noted. In the primary and larger haemorrhagic lesions there is free haemorrhage from the alveolar capillaries, while the alveolar ducts and bronchioles are air-containing and almost free from haemorrhage. The larger bronchioles and small bronchi give good evidence of compression of the lung from without. They are collapsed, and practically all the goblet cells in the mucosa have been ruptured, causing a stream of mucus to flow into the bronchial lumen; with the mucus there is a little blood but very little air. Incidentally this shows well how much mucus is available from goblet cells alone to make the bronchial mucous plugs of the influenza lung. The second main type is the result of inhaled blood, and is apt to be mixed with the previous type. The haemorrhagic lesions themselves are not necessarily fatal. Rapid death is due to shock. The probable reasons for more severe lung lesions are the severity of the causative factor and the state of the patient. Closeness to the bomb when in the open, and being caught in a building near a bomb explosion, are the causative factors. The great majority of cases so far reported have come from bombed houses and shelters; pulmonary concussion appears to be infrequent in cases injured in the open. By the state of the patient is meant whether they are obese or not, whether they have flexible or rigid chest walls, whether their lungs are in the position of inspiration (making it worse) or expiration and the amount of clothing worn. A very important factor in bringing about a compressible body of air is the involuntary spasm

of the glottis invoked by the feeling of danger. —*Pulmonary Concussion (Blast)*, *G. R. Osborn, Brit. M. J., April 5, 1941, 1: 506.*—(*D. H. C.*)

Hernia of Lung.—Hernia of the lung is defined as a protrusion of the lung and its pleural covering beyond its usual boundaries. Prolapse of the lung through a ruptured parietal pleura differs from it in that there is no hernial sac. Pulmonary hernias are classified into congenital and acquired types on an aetiological basis. The former occur primarily because of anomalies of development with defects of the bony or soft tissue structures of the chest wall. Acquired types are more common. They may be traumatic, spontaneous or pathological in origin. The traumatic is the most frequent type of lung hernia; prolonged steady pressure of the expanding lung or sudden increased pressure as from violent exertion may cause the traumatized part to give way and permit herniation. Spontaneous hernia results from lowered resistance in the thoracic wall which is due to developmental factors plus an abnormal or prolonged increase in the air pressure of the lung. It occurs most frequently in such areas of anatomical weakness as the superior aperture of the thoracic cage and in the intercostal spaces in the anterior chest wall medial to the costochondral junction of the ribs. Such hernias are seen in patients with chronic cough, chronic severe pulmonary emphysema or in individuals subjected to short but severe periods of exertion associated with extreme changes in intrathoracic pressure. In acquired hernias of pathological origin the thoracic wall has been weakened by some destructive process such as abscess of the chest wall, rib caries or malignant disease. According to location, hernias are classified into (*1*) diaphragmatic, (*2*) thoracic or intercostal and (*3*) cervical. Mediastinal hernias are excluded from this list, although considered by some to be pulmonary hernias. Herniation of the lung through the diaphragm is extremely unlikely because of the pressure relationships involved. The cervical type of hernia is uncommon; these occur between the scalenus anticus and the sternomastoid muscles, an anatomical area where the lung is least protected by bone and muscle. The protrusion usually presents itself in front of the neck at one side of the larynx. The great majority of pulmonary hernias, excluding the mediastinal ones, belong to the thoracic or intercostal group. They occur near the sternum internal to the costochondral junction, where the intercostal musculature is thinnest, or near the vertebrae at a weak point internal to the rib angle where the internal intercostal muscle ends. A case report is made of a patient exhibiting an acquired traumatic intercostal type of lung hernia. The hernia appeared about one month following an injury to the lower right anterior chest and was detected clinically by the appearance of a bulge during forced expiration with the glottis closed (Valsalva experiment). Roentgen examination during quiet respiration showed no abnormality. However, during the performance of the Valsalva act, a localized area of radiolucency was visible just above the right diaphragm in the sixth right intercostal space anteriorly near the lateral chest wall.—*Hernia of the Lung, A. Hartung & J. W. Grossman, Am. J. Roentgenol., September, 1941, 46: 321.*—(*W. H. M.*)

Clubbing of Fingers.—A rare case of unilateral clubbing of the fingers is described. A male, aged fifty-four, was admitted to the hospital complaining of pain in the right arm and a lump on the right side of the neck for six months. Examination revealed a pulsatile swelling two and one-half by two and three-quarter inches just above the right clavicle; a systolic thrill and murmur were present over the swelling. There was pitting oedema and venous engorgement of the right upper limb with pronounced finger clubbing of the right hand and curvature of the nails. The left extremity was normal. The Wassermann reaction was strongly positive. The case was diagnosed as syphylitic aortitis, with right subclavian aneurysm and secondary clubbing of the fingers of the right hand. Treatment

prescribed was rest, potassium iodide, mercury and bismuth. Under this therapy the aneurysm shrunk to about three-quarters of its original size and the oedema disappeared. However, the patient, when seen six months later, had voluntarily abandoned treatment for three months: his aneurysm was much larger and the clubbing more pronounced. Of the 29 cases of unilateral clubbing of the fingers reported in the literature, 24 occurred with aneurysms and in the remaining 5, pressure was observed on the vessels and nerves of the affected limb. In 2 of the reported cases the clubbing disappeared wholly or partially after operation on the aneurysm. Pressure is the common factor in these unilateral cases, but whether it was on the arteries, veins, nerves or lymphatics or a combination of these, is not clear, as the clinical signs accompanying the clubbing varied considerably. It is suggested that local circulatory embarrassment due to pressure causes anoxaemia which in turn brings about clubbing.—*A Case of Unilateral Clubbing of the Fingers with a Summary of the Literature, R. E. Rodgers, Brit. M. J., September 27, 1941, 2: 439.*—(*D. H. C.*)

Cancer of Lung.—A case is reported of a seventy-five year old Negro whose complaints included dyspnoea, cough, weakness and loss of weight. A small hard nodule was present near the right nipple. Roentgen examination of the chest revealed opacity of the right side, with mediastinal shift to the left. Following a rise in temperature to 105.2 F. accompanied by rapid respiration and cyanosis, a thoracocentesis was performed and a considerable amount of black-brown fluid was removed from the right chest. The patient died after six days. At autopsy, the entire right lung was seen to be liquefied down to the hilar structures, and the pleural space was filled with black fluid and autolyzed lung having only a slight odor. The right branch of the pulmonary artery was dilated and occluded by a large thrombus. Microscopical examination of these areas including the thrombus revealed invasion with carcinoma cells. The left lung was free of tumor. This is believed to be the first record of such a complication following bronchogenic carcinoma.—*Obturator Thrombosis of the Right Pulmonary Artery and Necrosis of the Entire Lung with Hilar Bronchogenic Carcinoma, E. G. Bahrents, Arch. Path., February, 1942, 33: 259.*—(*D. G. F.*)

Tumor of Diaphragm.—A case is reported of a seventy-three year old woman, admitted to the hospital with a history of swelling of the feet for three weeks, followed by weakness, anorexia and occasional vomiting, of frequent hiccup for a week, and of unusual thirst for four days. On admission, the area over the base of the right lung posteriorly was flat to percussion, and there was no transmission of voice or breath sounds: over the left base there were dulness and diminished breath sounds. The abdominal wall was oedematous, as were the back and lower extremities. Roentgenograms revealed what appeared to be a small amount of fluid in the right chest; the stomach and intestines showed hypermotility, and the duodenal cap filled poorly. The urine contained some albumin, red cells and casts. The patient eventually became stuporous, swelling and cyanosis of the right hand developed on the next day and death followed. At autopsy, a large fibrosarcoma was found in the region of the right crus of the diaphragm; the mass was fused with the lower lobe of the right lung and the posterior aspect of the liver, surrounded the diaphragmatic portion of the inferior vena cava, and enveloped the right adrenal gland. The inferior vena cava was thrombosed below its entrance into the right atrium and thrombus extended backward into the hepatic veins. The tumor displaced but did not surround the major branches of the upper abdominal aorta, as well as the right renal vessels. There was some fluid in the right pleural cavity and patchy pneumonia was present in the lungs. The position of the tumor and its microscopical appearance point to its origin in the connective tissue elements of the diaphragm, probably the tendenous centre; the irregularity of

the cellular arrangement and size, together with the tendency of the tumor to invade locally, indicate its malignancy.—*Fibrosarcoma of the Diaphragm, Report of a Case with Review of the Literature, M. A. Hyman & M. Lederer, Arch. Path., February, 1941, 31: 204.*—(*D. G. F.*)

Intrathoracic Neurogenic Tumors.—Attention is called to the frequency, variability and recognition of intrathoracic neurogenic tumors. Seven such tumors and one metastatic lesion resembling a neurogenic tumor are reported, the clinical manifestations are enumerated and the means of diagnosis is discussed. The importance of roentgen examination is emphasized and the roentgen characteristics are described.—*Intrathoracic Neurogenic Tumors, K. Kornblum & H. H. Bradshaw, Radiology, October, 1941, 37: 391.*—(*G. F. M.*)

Hodgkin's Disease.—Gordon tests performed with lymph nodes from 21 cases of Hodgkin's disease diagnosed microscopically were positive in 16, or 76.2 per cent. A higher percentage of positive results was obtained with biopsy specimens than with tissue obtained at necropsy; the latter may in part have been due to the greater amount of fibrosis and necrosis usually present in the nodes of advanced cases. In most instances, two or three rabbits were used for each specimen. Nodes of 40 control cases, including disease types from which Hodgkin's disease must be differentiated, such as lymphosarcoma, leucaemia and non-specific and tuberculous lymph node involvement, all gave negative reactions. A study of 8 false positive reactions reported in the literature revealed that most of these cases could have been differentiated from Hodgkin's disease microscopically. The reported presence of the Gordon agent in many tissues, including bone marrow, leucocytes and human spleen, indicates that it is nonspecific, although the test serves as a reliable criterion in the differential diagnosis of lymph node disease is accompanied by histological examination. The properties of the agent make it most likely that it is non-living, probably enzymatic in character.—*Reliability and Significance of the Gordon Test in Hodgkin's Disease, P. E. Steiner, Arch. Path., January, 1941, 31: 1.*—(*D. G. F.*)

Spontaneous Pneumothorax.—Spontaneous pneumothorax in apparently healthy individuals is not a particularly rare occurrence. During the past twenty years, 58 cases have been seen in private practice. In none of these was there any evidence of underlying pulmonary disease. In the literature of previous years there are a few reports of autopsy records of patients who have died with spontaneous pneumothorax. These suggest that the rupture of an emphysematous bleb has been the cause of the pneumothorax. The emphysema that is observed in them is not the ordinary variety of alveolar distension but is really subpleural blebs without alveolar disease. The production of the blebs and of the spontaneous pneumothorax caused by rupture of one of these has been a matter of some discussion. In a normal person before any severe exertion is made a deep inspiration is taken. At the peak of exertion the glottis is closed and the thorax compressed. The diaphragm becomes fixed in the position of deep inspiration and air is forced out of the lower lobes into the upper lobes, distending these latter to the utmost. Roentgenograms of apparently normal individuals taken in inspiration and expiration and in deep inspiration with the glottis closed and the thorax compressed show distension and increased aeration of the upper lobes of the lungs. This forceful inflation can easily overdistend and rupture the elastic fibres in the subpleural alveoli of the upper lobes. This results in the development of emphysematous blebs. In most such blebs the air can easily be expelled by manual pressure when the lungs have been removed from the body, but in some the reduction of the blebs is difficult. This is to be explained by a check valve mechanism which allows air to enter the bleb but does not allow it to escape. The enlargement and thinning of the wall of the bleb

continues until rupture of the wall takes place, thereby releasing air into the pleural cavity. Tuberculosis has often been thought to be a primary cause of spontaneous pneumothorax but this is not true in all cases. Fifteen of the present series of 58 cases were tested by intracutaneous tuberculin tests and 9 of these were found to be negative reactors. That spontaneous pneumothorax occurs in nontuberculous lungs is also supported by the fact that the collapsed lung usually is entirely free from the chest wall, whereas it might be expected that adhesions would be present if tuberculosis were a cause. In only 3 of this series of 58 cases were there any adhesions between the visceral and parietal pleural layers. In each of the 3 cases in which adhesions were present tuberculosis developed about two years later. Among these 58 patients only 2 were females. The average age was twenty-eight years. The youngest patient was fifteen years of age and the oldest fifty-six. The patients in general were healthy and robust individuals. Seventeen patients in this group could not associate the attack with any activity of theirs at the time of its occurrence. Eighteen other patients stated that there was no active exertion before the occurrence of the spontaneous pneumothorax. Twenty-three patients distinctly recalled severe exertion which shortly preceded the occurrence of the spontaneous pneumothorax. If exertion is a cause it is only a contributory one. The emphysematous bleb must already be present and communicating with the bronchus by a check valve mechanism. The exertion only causes a small increase in the amount of air within the bleb and probably only occasionally is a direct cause of the rupture. Many of the patients in this present series were engaged in sedentary occupations or in those involving little exertion. The two outstanding symptoms of spontaneous pneumothorax are thoracic pain and dyspnoea. The pain is sudden in origin and usually so sharp and severe that the patient is greatly distressed. In only 8 cases was the pain of secondary importance. The 50 patients with severe pain found relief when they were resting in bed. Dyspnoea became progressively worse until the patients were placed on absolute rest. In most cases the shortness of breath gradually disappeared after forty-eight hours of bed-rest. The dyspnoea is not entirely due to collapse of the lung. At the beginning of the attack the pain may be so excruciating that the entire thorax is immobilized and breathing becomes rapid and shallow. With partial collapse of the lung blood flows through a poorly ventilated lung and this incompletely aerated venous blood is then returned to the left auricle. Pollution of the arterial blood is an important factor in the dyspnoea and discomfort of the patients and is also responsible for the rapid pulse rate observed in the first twenty-four hours. A further cause of dyspnoea is the loss of respiratory capacity of the lung because of pneumothorax. Only 3 of the 58 patients had fever. Cough occurred in 16 but was not severe and usually not productive. At the moment of the attack the patient is almost in shock, respirations are increased in rate and the pulse is rapid. Roentgenograms show varying amounts of collapse of the lung. In many cases, because of the small amount of air present within the thorax, the pneumothorax may be overlooked on roentgenograms taken in deep inspiration, whereas it would be much more evident if the roentgenograms were made in expiration. In most instances there is a selective collapse of the upper lobe. Small pleural effusions occurred in 22 of the 58 cases. Among the 58 cases the pneumothorax was small in 41, moderate in size in 50, large in 3 and complete in 9 cases. Reëxpansion of the lung depends on the closure of the pleural opening and the amount of surface of the lung available for absorbing the gases in the pleural cavity. Absorption of the air is rapid and the lung may reëxpand completely in from one to four weeks. When the pneumothorax is large it is wise to aspirate air to facilitate rapid reexpansion. In 41 of the cases of this series only one attack occurred. Recurrences occurred in 17 cases; of this number 9 had 2 attacks, 2 had 3, 2 had 4, 2 had 5, and 2 had 8

attacks. The prognosis is usually very good, although 2 of the patients in this series developed tuberculosis two years after the occurrence of the spontaneous pneumothorax. In treating the patients with spontaneous pneumothorax bed-rest for from four to six weeks is usually sufficient. In patients with complete or almost complete collapse of the lung aspiration of air from within the pleural cavity is often necessary. Aspiration should not be done during the first three days unless the patient complains of marked dyspnoea and discomfort. The production of highly negative intrapleural pressure by means of aspiration of air may reopen a healing wound in the visceral pleura. Once the diagnosis of spontaneous pneumothorax has been made, the patient must be educated never to close his glottis if he can avoid it. When such patients have to indulge in hard exertion it is wise for them to keep talking during this period in order to keep the glottis open.—*Spontaneous Pneumothorax in Apparently Healthy Individuals, G. G. Ornstein & L. Lercher, Quart. Bull. Sea View Hosp., April, 1942, 7: 149.*—(*A. A. E.*)

Chyliform Pleural Effusions.—A man of forty-one years was hospitalized with a three-day story of sudden pain in the left chest and dyspnoea, followed by a temperature of 41.5°C. A massive pleural effusion, displacing the mediastinum, was found and 1.5 liters of serofibrinous fluid was removed. The rapid reaccumulation of fluid required repeated aspiration. One day, without apparent cause, the effusion was found to have become chyliform. There were no neoplastic cells. Continuous subcutaneous drainage was instituted without relief of the symptoms of mediastinal displacement, because drainage was very slow as compared with cases of serofibrinous effusion. On the assumption that this was due to the density of the fluid, wider needles were used, without success. Measurement of the intrapleural pressure showed that despite the large effusion, it was negative, and consequently did not force the fluid through the needle. X-ray examination showed the right lung almost completely collapsed and enclosed in a thick shell, which not only did not permit it to expand normally but impeded the the circulation of blood and lymph because of the severe and extensive adhesive process which involved the mediastinum, already displaced by the effusion. The negative pressure was attributed to loss of pulmonary elasticity due to either atelectasis or fibrosis. A Potain aspiration, carried out in an attempt to drain the pleural cavity as completely as possible, was stopped because of pain and tachycardia. Nevertheless, X-ray examination showed partial reëxpansion of the lung and shift of the mediastinum to the right. From this point on fluid reaccumulated more slowly and became serofibrinous. After a second Potain aspiration, the patient's condition showed definite improvement and progressive reëxpansion of the lung occurred. Further therapy consisted of artificial respiration in the iron lung and the use of blow-bottles. Since Vernage's description of a milky effusion in 1700, there have been many studies of such effusions. They need not always be due to a lesion of the chyle-bearing vessels although such lesions have been demonstrated in some cases at autopsy. Extrinsic or intrinsic occlusion of the thoracic duct is sufficient to cause such effusions through dilatation and rupture of the afferent lymphatics, especially when an inflammatory or neoplastic process blocks the retrograde lymphatic circulation as well. The theory that chyliform effusions are due to fatty degeneration of leucocytes does not explain those originating as hemorrhagic or serofibrinous processes. Furthermore, chemical study has shown in some cases not an excess of fats but a large quantity of metalbumin as the cause of turbidity. Truly chylous effusions are almost always traumatic in origin and the fluid has a typical character. This and the grave prognosis distinguish them from chyliform effusions. Chylous effusions show coagula and clear completely on addition of Adam's fluid; chyliform fluid shows no coagula and clears only partially. In the case presented, the chyliform effusion is attributed to the

mediastinal displacement interfering with normal lymphatic circulation and causing rupture of some of the pleural lymphatics in a subject predisposed by long-standing pleural inflammation and consequent damage to the lymphatics. The absence of the characteristics of chylous effusions, which would be expected with ruptured lymphatics, is attributed to the small quantity of chyle present in the fluid. Actually, after a fatty meal, the fluid not only became thicker, but also, on standing overnight, coagulated to some extent. By exclusion, because of its benign course, the case reported is considered tuberculous.—*Contribución al estudio de la fisiopatogenia de las pleuresías inagotables y quiliformes, N. Romano, Rev. Asoc. med. argent., November 15–30, 1941, 40: 866.*—(*H. B. S.*)

Rheumatic Pleurisy.—A twelve year old boy developed pleurisy with effusion several weeks after he had had rheumatic polyarthritis, rheumatic myocarditis, mitral regurgitation. A healed Ghon tubercle could be seen, the tuberculin test was positive. No organisms could be found in the pleural fluid. The exudate was resorbed within a short time. Manifestations of rheumatic fever are: arthritis in 70 per cent of the cases, chorea minor in 50 per cent, subcutaneous fibroid nodules in 14 per cent, pericarditis, myocarditis, endocarditis, or pancarditis in 18 per cent, skin manifestations in 12 per cent.—*Pleurisy with Effusion as a Manifestation of Rheumatic Fever, Arild E. Hansen, Journal-Lancet, May, 1942, 62: 158.*—(*G. C. L.*)

Leprosy with Tuberculosis.—A case of a forty-four year old Bolivian Indian male with nodular leprosy is reported. Symptoms included weight loss, cough, aphonia, backache and diarrhoea, with increasing weakness, dyspnoea and fever preceding death. At autopsy there were papular crusted lesions on the face. The epiglottis and the mucosa of the larynx and trachea were thickened, and microscopical examination revealed leprous granulation tissue containing characteristic Virchow foamy cells filled with numerous acid-fast bacilli. The lungs contained numerous miliary tubercles, together with groups of foamy cells; the kidneys showed scattered tubercles. The liver and spleen were enlarged, containing numerous large areas of caseation surrounded by typical tuberculous granulation tissue containing relatively few acid-fast bacilli; in addition there were numerous small leprous foci in the liver, while foamy cells were scattered through the splenic pulp. Local lymph nodes were also involved. Circular ulcers were present in the large bowel; these ulcers contained Virchow cells, and similar cells were present in other portions of the colon as well. Leprous ulcerative colitis seems to be the most frequent cause of death in patients with leprosy. Caries was present in the fifth and eleventh dorsal vertebrae, with an epidural abscess at the level of the latter. The presence of large tuberculomata in the liver, spleen and involved lymph nodes may be due to primary infection since tuberculosis has only recently spread to the Indian race in Bolivia. Lepromata of the inner organs do not exceed pea-size.—*Leprosy with Widespread Tumor-like Tuberculosis, F. Wenger, Arch. Path., July, 1941, 32: 112.*—(*D. G. F.*)

Disseminated Lupus Erythematosus.—An anatomical study of 20 consecutive cases of disseminated lupus erythematosus indicates its morphological basis to be a widespread progressive degeneration of collagen with characteristic local expression in specific organs, such as the heart and kidneys. Involvement of the serous membranes is usual but varies in intensity. The pericardium may show no gross change, or, at the other extreme, may be partially or completely obliterated by fibrous adhesions or a thick gelatinous tissue. Histologically, nearly every case shows fibrosis, capillarization and cellular infiltration; in addition, distinctive changes are frequently seen in the epicardium, the ground substance between the collagen fibres appearing altered and deeply eosinophilic while the fibres themselves lose their wavy appearance and appear irregularly thickened and refractile (fibrinoid

degeneration). This is accompanied by proliferation of fibroblasts often showing degenerative changes. In the gelatinous adherent pericardium there is obliteration of the sharp contrast between fibres and ground substance; the latter increases in bulk and appears "collagenized." Pleuritis, perisplenitis and perihepatitis are frequent and fibrinoid degeneration may be seen. Endocardial vegetations were evident grossly in 40 per cent and microscopically in an additional 20 per cent of the cases. When well developed, they are pathognomonic. They occur in the valves, but the mural endocardium or chorda tendineae may also be involved. The vegetations consist of altered valve tissue, the ground substance becoming homogeneous and eosinophilic, increasing in mass and elevating the endothelium; the latter may show degenerative changes. The collagen fibres in the deep layers of the endocardium may show changes sometimes progressing to fragmentation, together with associated cellular changes which may progress to necrosis. Capillarization appears late and is not extensive; fibrosis occurs, but the new fibrous tissue may also undergo fibrinoid degeneration. Superimposed bacterial implantation occurred in 20 per cent of the cases. Characteristic alteration of the myocardial connective tissue occurred in 35 per cent. The lungs usually exhibit a nonspecific bronchopneumonia. The liver shows no characteristic alteration, nor does the spleen, except for a periarterial fibrosis limited to the central and periciliary arteries apparently caused by collagenization of the adventitial reticulum fibres. These sclerotic rings may far exceed normal trabeculae in thickness and may represent another type of response of the collagen-reticulum system to the injurious factor; they were present in 19 out of 20 cases. The kidneys show a peculiar alteration of the basement membranes of the glomerular loops; they appear thickened and rigid, resembling wire loops and are deeply eosinophilic, resembling amyloid. This is probably due to a localized fibrinoid degeneration of the ground substance. The swollen basement membrane may intrude into the capillary lumen, giving rise to so-called "hyaline thrombi." All the loops are not necessarily involved, nor are all the glomeruli. Some appear fragmented, others necrotic; in 25 per cent of the cases necrosis was present in the absence of wire loop change. Wire loop change and necrosis appear to be different grades of the same alteration depending on the intensity of the underlying injury. Lymph nodes may show necrosis, together with characteristic collagen alterations in septa and capsules. Vascular lesions, especially frequent in the kidneys may be present in any organ; sometimes, as in the gastrointestinal tract they may be the only manifestation of collagen injury. The lesions vary in degree from fibrinoid changes in the intima, often narrowing the vessel lumen, to more extensive involvement involving media and adventitia, with fusion of the masses and complete blockage of the lumen. With complete fibrinoid change, the muscle and elastic elements are also destroyed. Occasionally, massive necrosis of the vessel wall, not associated with fibrinoid, is seen. Thrombosis is rare. The veins are affected infrequently. Characteristic changes are frequently present in the skin, chiefly in the upper layers of the corium, and in the more advanced lesions there is destruction of the collagen fibres with considerable degeneration of the cellular elements. Capillaries are dilated and there is extravasation of red cells, although the capillary endothelium remains intact; these changes are present even in the absence of definite collagen alteration. In general, the compactness of the connective tissue of the corium makes the collagen injury less obvious in the skin than elsewhere, or than the degeneration and necrosis of the fibroblasts. Lupus erythematosus differs from other diseases involving widespread collagen injury. In rheumatic fever this injury is less intense and accompanied by actively proliferating Aschoff cells; the proliferation of fibroblasts is progressive and does not show the degenerative changes seen in pupus. The degeneration and necrosis of collagen in periarteritis nodosa is accompanied by definite inflammatory reaction with eosino-

philic infiltration. In addition, the vessels of larger calibre suffer greatest damage, whereas in lupus the smaller arteries and capillaries are predominantly affected. Unlike lupus too, periarteritis is usually associated with massive destruction of the vessel wall with haemorrhage or aneurysm formation and thrombosis is frequent. In glomerulonephritis, exudative and proliferative changes in the kidneys overshadow alterative changes; the wire loops are occasionally demonstrable but are associated with characteristic glomerular changes, and collagen changes are absent from other organs. The loop thickening in eclampsia does not resemble that seen in lupus but is rather due to fusion of loops due to epithelial cell degeneration. Amyloid is readily differentiated by staining. Hyalinization of loops in the arteriosclerotic kidney can be differentiated by staining and by general gross and microscopical appearance. The end results of fully developed vascular necrosis in malignant nephrosclerosis and lupus may be indistinguishable, but the morphogenesis is totally different. Active tuberculosis was absent in the cases studied, and arrested tuberculosis was infrequent; the disease is therefore not likely to be an unusual reaction to tuberculous infection. Lupus erythematosus differs from all these diseases in that it represents a definite disturbance of collagen affecting all organs and tissues, essentially degenerative in character, in the absence of a palpable injurious agent. Such widespread characteristic alteration is seen in no other disease except scleroderma.—*Pathology of Disseminated Lupus Erythematosus, P. Klemperer, A. D. Pollack & G. Baehr, Arch. Path., October, 1941, 32: 569.*—(*D. G. F.*)

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